AMENDMENT TO THE SPECIFICATION

Please make the following amendments to the specification.

Delete paragraphs [0009] to [0035] of the Summary on Invention and replace them with the following paragraphs.

The present invention is directed to an isolated nucleic acid wherein the sequence of the nucleic acid consists of the sequence of SEQ ID NO: 1917, or the complement of SEQ ID NO: 1917 or the RNA equivalent of SEQ ID NO: 1917, wherein the complement is identical in length to the nucleic acid of (a) or (b). SEQ ID NO: 1917 is the viral hairpin sequence of the Viral Genomic Address Messenger 1931.

The present invention is also directed to an isolated nucleic acid wherein the sequence of the nucleic acid consists of the sequence of SEQ ID NO: 4642, an RNA equivalent of SEQ ID NO: 4642, or the complement of SEQ ID NO: 4642 or the RNA equivalent of SEQ ID NO: 4642, wherein the complement is identical in length to the nucleic acid of (a) or (b). SEQ ID NO: 4642 is the viral miR of the viral hairpin sequence as set forth in SEQ ID NO: 1917 of the Viral Genomic Address Messenger 1931 (VGAM1931) and modulates expression of host target genes thereof wherein the function and utility of the host genes is known in the art.

The present invention is also directed to a vector comprising the nucleic acid wherein the sequence of the nucleic acid consists of the sequence of SEQ ID NO: 4642, an RNA equivalent of SEQ ID NO: 4642, or the complement of SEQ ID NO: 4642 or the RNA equivalent of SEQ ID NO: 4642, wherein the complement is identical in length to the nucleic acid of (a) or (b). The present invention is also directed to a vector comprising the nucleic acid wherein the sequence of the nucleic acid consists of the sequence of SEQ ID NO: 1917, an RNA equivalent of SEQ ID NO: 1917, or the complement of SEQ ID NO: 1917or the RNA equivalent of SEQ ID NO: 1917, wherein the complement is identical in length to the nucleic acid of (a) or (b).

The present invention is also directed to an isolated nucleic acid wherein the sequence of the nucleic acid consists of the sequence of SEQ ID NO: 4642, an RNA equivalent of SEQ ID NO: 4642, or the complement of SEQ ID NO: 4642 or the RNA equivalent of SEQ ID NO: 4642, wherein the complement is identical in length to the nucleic acid of (a) or (b). SEQ ID NO: 4642 is the viral miR of the viral hairpin sequence as set forth in SEQ ID NO: 1917 of the Viral Genomic Address Messenger 1931 (VGAM1931) and modulates expression of host target genes thereof wherein the function and utility of the host genes is known in the art.

The present invention is also directed to a probe comprising the nucleic acid wherein the sequence of the nucleic acid consists of the sequence of

SEQ ID NO: 4642, an RNA equivalent of SEQ ID NO: 4642, or the complement of SEQ ID NO: 4642 or the RNA equivalent of SEQ ID NO: 4642, wherein the complement is identical in length to the nucleic acid of (a) or (b). The present invention is also directed to a probe comprising the nucleic acid wherein the sequence of the nucleic acid consists of the sequence of SEQ ID NO: 1917, an RNA equivalent of SEQ ID NO: 1917, or the complement of SEQ ID NO: 1917 or the RNA equivalent of SEQ ID NO: 1917, wherein the complement is identical in length to the nucleic acid of (a) or (b).

Amend paragraphs [0059] and [0060] as follows:

~~Figs. 15A through 2739D are~~ Figures 15A-D are schematic diagrams illustrating sequences, functions and utilities of ~~2725 specific viral genes of the novel group of viral regulatory genes of the~~the VGAM 1931 gene expressing the hairpin as set forth in SEQ ID NO: 1917 and the miRNAs set forth in SEQ ID NO: 4642~~present invention~~, which were detected using the bioinformatic gene detection system described hereinabove with reference to Figs. 1 through 8; and

~~Figs. 2740 through 3297 are schematic diagrams illustrating sequences, functions and utilities of 558 specific viral genes of a group of novel regulatory "operon like" viral genes of the present invention, detected using the bioinformatic gene detection system described herein above with reference to Figs. 9 through 14.~~

After the heading "DETAILED DESCRIPTION," and before paragraph [0062], add the following paragraphs:

The present invention relates to a novel group of bioinformatically detectable, viral regulatory RNA genes, which repress expression of host target host genes, by means of complementary hybridization to binding sites in untranslated regions of these host target host genes. It is believed that this novel group of viral genes represent a pervasive viral mechanism of attacking hosts, and that therefore knowledge of this novel group of viral genes may be useful in preventing and treating viral diseases.

In various preferred embodiments, the present invention seeks to provide improved method and system for detection and prevention of viral disease, which is mediated by this group of novel viral genes.

Accordingly, the invention provides several substantially pure nucleic acids (e.g., genomic nucleic acid, cDNA or synthetic nucleic acid) each encoding a novel viral gene of the VGAM group of gene, vectors comprising the nucleic acids , probes comprising the nucleic acids , a method and system for selectively modulating translation of known "target" genes utilizing the vectors, and a method and system for detecting expression of known "target" genes utilizing the probe.

By "substantially pure nucleic acid" is meant nucleic acid that is free of the genes which, in the naturally-occurring genome of the organism from which the nucleic acid of the invention is derived, flank the genes discovered and isolated by the present invention. The term therefore includes, for example, a recombinant nucleic acid which is incorporated into a vector, into an autonomously repl icating plasmid or virus, or into the genomic nucleic acid of a prokaryote or eukaryote at a site other than its natural site; or which exists as a separate molecule (e.g., a cDNA or a genomic or cDNA fragment produced by PCR or restriction endonuclease digestion) independent of other sequences. It also includes a recombinant nucleic acid which is part of a hybrid gene encoding additional polypeptide sequence.

"Inhibiting translation" is defined as the ability to prevent synthesis of a specific protein encoded by a respective gene, by means of inhibiting the translation of the mRNA of this gene. "Translation inhibiter site" is defined as the minimal nucleic acid sequence sufficient to inhibit translation.

There is thus provided in accordance with a preferred embodiment of the present invention a bioinformatically detectable novel viral gene encoding substantially pure nucleic acid wherein: RNA encoded by the bioinformatically detectable novel viral gene is about 18 to about 24 nucleotides in length, and originates from an RNA precursor, which RNA precursor is about 50 to about 120 nucleotides in length, a nucleotide sequence of a first half of the RNA precursor is a partial inversed-reversed sequence of a nucleotide sequence of a second half thereof, a nucleotide sequence of the RNA encoded by the novel viral gene is a partial inversed-reversed sequence of a nucleotide sequence of a binding site associated with at least one host target gene, and a function of the novel viral gene is bioinformatically deducible.

There is further provided in accordance with another preferred embodiment of the present invention a method for anti-viral treatment comprising neutralizing said RNA.

Further in accordance with a preferred embodiment of the present invention the neutralizing comprises: synthesizing a complementary nucleic acid molecule, a nucleic sequence of which complementary nucleic acid molecule is a partial inversed-reversed sequence of said RNA, and transfecting host cells with the complementary nucleic acid molecule, thereby complementarily binding said RNA.

Further in accordance with a preferred embodiment of the present invention the neutralizing comprises immunologically neutralizing.

There is still further provided in accordance with another preferred embodiment of the present invention a bioinformatically detectable novel viral gene encoding substantially pure nucleic acid wherein: RNA encoded by the bioinformatically detectable novel viral gene includes a plurality of RNA sections, each of the RNA sections being about 50 to about 120 nucleotides in length, and including an RNA segment, which RNA segment is about 18 to about 24 nucleotides in length, a nucleotide sequence of a first half of each of the RNA sections encoded by the novel

viral gene is a partial inversed-reversed sequence of nucleotide sequence of a second half thereof, a nucleotide sequence of each of the RNA segments encoded by the novel viral gene is a partial inversed-reversed sequence of the nucleotide sequence of a binding site associated with at least one target host gene, and a function of the novel viral gene is bioinformatically deducible from the following data elements: the nucleotide sequence of the RNA encoded by the novel viral gene, a nucleotide sequence of the at least one target host gene, and function of the at least one target host gene.

Further in accordance with a preferred embodiment of the present invention the function of the novel viral gene is bioinformatically deducible from the following data elements: the nucleotide sequence of the RNA encoded by the bioinformatically detectable novel viral gene, a nucleotide sequence of the at least one target host gene, and a function of the at least one target host gene.

Still further in accordance with a preferred embodiment of the present invention the RNA encoded by the novel viral gene complementarily binds the binding site associated with the at least one target host gene, thereby modulating expression of the at least one target host gene.

Additionally in accordance with a preferred embodiment of the present invention the binding site associated with at least one target host gene is located in an untranslated region of RNA encoded by the at least one target host gene.

Moreover in accordance with a preferred embodiment of the present invention the function of the novel viral gene is selective inhibition of translation of the at least one target host gene, which selective inhibition includes complementary hybridization of the RNA encoded by the novel viral gene to the binding site.

Further in accordance with a preferred embodiment of the present invention the invention includes a vector including the DNA.

Still further in accordance with a preferred embodiment of the present invention the invention includes a method of selectively inhibiting translation of at least one gene, including introducing the vector.

Moreover in accordance with a preferred embodiment of the present invention the introducing includes utilizing RNAi pathway.

Additionally in accordance with a preferred embodiment of the present invention the invention includes a gene expression inhibition system including: the vector, and a vector inserter, functional to insert the vector into a cell, thereby selectively inhibiting translation of at least one gene.

Further in accordance with a preferred embodiment of the present invention the invention includes a probe including the DNA.

Still further in accordance with a preferred embodiment of the present invention the invention includes a method of selectively detecting expression of at least one gene, including using the probe.

Additionally in accordance with a preferred embodiment of the present invention the invention includes a gene expression detection system

including: the probe, and a gene expression detector functional to selectively detect expression of at least one gene.

Further in accordance with a preferred embodiment of the present invention the invention includes an anti-viral substance capable of neutralizing the RNA.

Still further in accordance with a preferred embodiment of the present invention the neutralizing includes complementarily binding the RNA.

Additionally in accordance with a preferred embodiment of the present invention the neutralizing includes immunologically neutralizing. Moreover in accordance with a preferred embodiment of the present invention the invention includes a method for anti-viral treatment including neutralizing the RNA.

Further in accordance with a preferred embodiment of the present invention the neutralizing includes: synthesizing a complementary nucleic acid molecule, a nucleic sequence of which complementary nucleic acid molecule is a partial inversed-reversed sequence of the RNA, and transfecting host cells with the complementary nucleic acid molecule, thereby complementarily binding the RNA.

Still further in accordance with a preferred embodiment of the present invention the neutralizing includes immunologically neutralizing.

Add the following paragraphs after paragraph [00161].

VGAM1931 RNA, herein schematically represented by VGAM2 binds complimentarily to a host target binding site located in an untranslated region of VGAM1931 host target RNA, herein schematically represented by VGAM2 HOST TARGET RNA, which host target binding site corresponds to a host target binding site such as BINDING SITE I, BINDING SITE II, or BINDING SITE III of Fig. 1, thereby inhibiting translation of VGAM1931 host target RNA, herein schematically represented by VGAM2 HOST TARGET RNA into VGAM1931 host target protein, herein schematically represented by VGAM2 HOST TARGET PROTEIN, both of Fig. 1.

Reference is now made to Fig. 15A, which is a simplified diagram providing a conceptual explanation of the mode by which a novel bioinformatically detected viral gene, referred to here as Viral Genomic Address Messenger 1931 (VGAM1931) modulates expression of host target genes thereof, the function and utility of which host target genes is known in the art.

VGAM1931 is a novel bioinformatically detected regulatory, non protein coding, viral micro RNA (miRNA) gene. The method by which VGAM1931 was detected is described hereinabove with reference to Figs. 2-8.

VGAM1931 GENE is a viral gene contained in the genome of Human herpesvirus 4. VGAM1931-HOST TARGET GENE is a human gene contained in the human genome.

VGAM1931 GENE encodes a VGAM1931 PRECURSOR RNA. Similar to other miRNA genes, and unlike most ordinary genes, the RNA transcribed by VGAM1931, VGAM1931 PRECURSOR RNA, does not encode a protein.

VGAM1931 PRECURSOR RNA folds onto itself, forming a 'hairpin structure' designated VGAM1931 FOLDED PRECURSOR RNA. As is well known in the art, this 'hairpin structure', is typical of RNA encoded by miRNA genes, and is due to the fact that the nucleotide sequence of the first half of the RNA encoded by a miRNA gene is an accurate or partial inversed-reversed sequence of the nucleotide sequence of the second half thereof. By "inversed-reversed" is meant a sequence which is reversed and wherein each nucleotide is replaced by a complementary nucleotide, as is well known in the art (e.g. ATGGC is the inversed-reversed sequence of GCCAT).

An enzyme complex designated DICER COMPLEX, 'dices' the VGAM1931 FOLDED PRECURSOR RNA into a single stranded ~22 nt long RNA segment, designated VGAM1931 RNA. As is known in the art, 'dicing' of a hairpin structured RNA precursor product into a short ~22nt RNA segment is catalyzed by an enzyme complex comprising an enzyme called Dicer together with other necessary proteins.

VGAM1931-HOST TARGET GENE encodes a corresponding messenger RNA, designated VGAM1931-HOST-TARGET RNA. VGAM1931-HOST-TARGET RNA comprises three regions, as is typical of mRNA of a protein coding gene: a 5' untranslated region, a protein coding region and a 3' untranslated region, designated 5'UTR, PROTEIN CODING and 3'UTR respectively.

VGAM1931 RNA binds complementarily to one or more host binding sites located in untranslated regions of VGAM1931-HOST-TARGET RNA. This complementary binding is due to the fact that the nucleotide sequence of VGAM1931 RNA is an accurate or a partial inversed-reversed sequence of the nucleotide sequence of each of the host binding sites. As an illustration, FIG. 1931A shows 3 such host binding sites, designated BINDING SITE-I, BINDING SITE-II and BINDING SITE-III respectively. It is appreciated that the number of host binding sites shown in FIG. 1931A is meant as an illustration only, and is not meant to be limiting – VGAM1931 may have a different number of binding sites in untranslated regions of a VGAM1931-HOST-TARGET RNA. It is further appreciated that while Fig. 15A depicts the host binding sites in the 3'UTR region, this is meant as an example only – the binding sites may be located in the 3'UTR region, the 5'UTR region, or in both 3'UTR and 5'UTR regions.

The complementary binding of VGAM1931 RNA to BINDING SITE-I, BINDING SITE-II and BINDING SITE-III inhibits translation of VGAM1931-HOST-TARGET RNA into VGAM1931-HOST-TARGET PROTEIN. VGAM1931-HOST-TARGET PROTEIN is therefore outlined by a broken line.

It is appreciated that VGAM1931-HOST-TARGET GENE in fact represents a plurality of host target genes of VGAM1931. The mRNA of

each of this plurality of host target genes of VGAM1931 comprises one or more host binding site, having a nucleotide sequence which is at least partly complementary to VGAM1931 RNA, and which when bound by VGAM1931 RNA causes inhibition of translation of one of a plurality of host target proteins of VGAM1931. Host target genes of VGAM1931 and their respective host binding sites, are described hereinbelow with reference to Fig. 15D.

It is further appreciated by one skilled in the art that the mode of translational inhibition illustrated by Fig. 15A with specific reference to translational inhibition exerted by VGAM1931 on one or more host target genes of VGAM1931, is in fact common to other known non-viral miRNA genes. As mentioned hereinabove with reference to the background section, although a specific complementary binding site has been demonstrated only for miRNA genes Lin-4 and Let-7, all other recently discovered miRNA genes are also believed by those skilled in the art to modulate expression of other genes by complementary binding, although specific complementary binding sites of these genes have not yet been found (Ruvkun G., 'Perspective: Glimpses of a tiny RNA world', Science 294 ,779 (2001)).

It is yet further appreciated that a function of VGAM1931 is inhibition of expression of host target genes, as part of a novel viral mechanism of attacking a host. Accordingly, utilities of VGAM1931 include diagnosis, prevention and treatment of viral infection by Human herpesvirus 4. Specific functions, and accordingly utilities, of VGAM1931 correlate with, and may be deduced from, the identity of the target genes which VGAM1931 binds and inhibits, and the function of these target genes, as elaborated hereinbelow with reference to Fig. 15D.

Reference is now made to Fig. 15B, which shows the nucleotide sequence of VGAM1931 PRECURSOR RNA of Fig. 15A, designated SEQ ID:1917, and a probable (over 74%) nucleotide sequence of VGAM1931 RNA of Fig. 15A, designated SEQ ID:4642. The nucleotide sequence of SEQ ID:4642 is marked by an underline within the sequence of VGAM1931 PRECURSOR RNA. Nucleotide sequence SEQ ID:1917 is located at position 151629 relative to the genome of Human herpesvirus 4.

Reference is now made to Fig. 15C, which shows the secondary folding of VGAM1931 PRECURSOR RNA, forming a 'hairpin structure' designated VGAM1931 FOLDED PRECURSOR RNA, both of Fig. 15A. The nucleotide sequence of SEQ ID:4642, which is highly likely (>74%) to be identical or highly similar to the nucleotide sequence of VGAM1931 RNA is marked on VGAM1931 FOLDED PRECURSOR RNA by a solid underline. It is appreciated that the complementary base-paring is not perfect, with 'bulges', as is well known in the art with respect to the RNA folding of all known miRNA genes.

Reference is now made to Fig. 15D, which is a table showing complementarity of host binding sites of VGAM1931, found in untranslated regions of host target genes of VGAM 1931, to SEQ ID:4642, which is highly likely (>74%) to be identical or highly similar

to the nucleotide sequence of VGAM1931 RNA of Fig. 15A. Each of the host binding sites described hereinbelow corresponds to a host binding site, such as BINDING SITE-I, BINDING SITE-II or BINDING SITE-III, all of Fig. 15A, and each of the host target genes of VGAM1931 described hereinbelow corresponds to VGAM HOST TARGET GENE of Fig. 15A.

As mentioned hereinabove with reference to Fig. 15A a function of VGAM1931 is inhibition of expression of host target genes, as part of a novel viral mechanism of attacking a host. Accordingly, utilities of VGAM1931 include diagnosis, prevention and treatment of viral infection by Human herpesvirus 4. It is appreciated that specific functions, and accordingly utilities, of VGAM1931 correlate with, and may be deduced from, the identity of the host target genes which VGAM1931 binds and inhibits, and the function of these host target genes, as elaborated herein below.

Reference is now made to COL6A1 BINDING SITE. collagen, type VI, alpha 1 (COL6A1, Accession NM_001848) is a host target gene of VGAM1931, corresponding to VGAM1931-HOST TARGET GENE of Fig. 15A. COL6A1 BINDING SITE is a host binding site found in the 3' untranslated region of COL6A1, corresponding to a host binding site such as BINDING SITE-I, BINDING SITE-II or BINDING SITE-III, all of Fig. 15A. Fig. 15D illustrates the complementarity of the nucleotide sequence of COL6A1 BINDING SITE, designated SEQ ID:7584, to the nucleotide sequence of VGAM1931 RNA of Fig. 15A, designated SEQ ID:4642.

A function of VGAM1931 is therefore inhibition of collagen, type VI, alpha 1 (COL6A1), a host gene which encodes a Protein that is associated with BETHLEM MYOPATHY, as part of a novel viral mechanism used by Human herpesvirus 4 for attacking a host. Accordingly, utilities of VGAM1931 include diagnosis, prevention and treatment of viral infection by Human herpesvirus 4. The function and utilities of COL6A1 have been established by previous studies, as described hereinabove with reference to Fig 1119D.

Reference is now made to SFRS1 BINDING SITE. splicing factor, arginine/serine-rich 1 (splicing factor 2, alternate splicing factor) (SFRS1, Accession NM_006924) is a host target gene of VGAM1931, corresponding to VGAM1931-HOST TARGET GENE of Fig. 15A. SFRS1 BINDING SITE is a host binding site found in the 3' untranslated region of SFRS1, corresponding to a host binding site such as BINDING SITE-I, BINDING SITE-II or BINDING SITE-III, all of Fig. 15A. Fig. 15D illustrates the complementarity of the nucleotide sequence of SFRS1 BINDING SITE, designated SEQ ID:13801, to the nucleotide sequence of VGAM1931 RNA of Fig. 15A, designated SEQ ID:4642.

Yet another function of VGAM1931 is therefore inhibition of splicing factor, arginine/serine-rich 1 (splicing factor 2, alternate splicing factor) (SFRS1), a host gene which encodes a Protein that plays an essential role in pre-mRNA splicing, as part of a novel viral mechanism used by Human herpesvirus 4 for attacking a host. Accordingly, utilities of

VGAM1931 include diagnosis, prevention and treatment of viral infection by Human herpesvirus 4. The function and utilities of SFRS1 have been established by previous studies, as described hereinabove with reference to Fig 323D. .

Reference is now made to HIP12 BINDING SITE. HIP12 (Accession XM_038791) is a host target gene of VGAM1931, corresponding to VGAM1931-HOST TARGET GENE of Fig. 15A. HIP12 BINDING SITE is a host binding site found in the 3' untranslated region of HIP12, corresponding to a host binding site such as BINDING SITE-I, BINDING SITE-II or BINDING SITE-III, all of Fig. 15A. Fig. 15D illustrates the complementarity of the nucleotide sequence of HIP12 BINDING SITE, designated SEQ ID:32922, to the nucleotide sequence of VGAM1931 RNA of Fig. 15A, designated SEQ ID:4642.

An additional function of VGAM1931 is therefore inhibition of (HIP12), a host gene which encodes a Protein that is a component of clathrin-coated pits and vesicles, that may link the endocytic machinery to the actin cytoskeleton., as part of a novel viral mechanism used by Human herpesvirus 4 for attacking a host. Accordingly, utilities of VGAM1931 include diagnosis, prevention and treatment of viral infection by Human herpesvirus 4.

The function of HIP12 has been established by previous studies. Huntingtin-interacting protein-1 (HIP1; 601767) is a membrane-associated protein that interacts with huntingtin (143100), the protein altered in Huntington disease. While attempting to isolate the mouse homolog of HIP1, Chopra et al. (2000) identified a homologous cDNA, which they designated Hip12. By screening a human frontal cortex cDNA library with an EST that showed homology to mouse Hip12, Chopra et al. (2000) cloned a full-length HIP12 cDNA encoding a deduced 1,068-amino acid protein that shares 47% sequence identity with HIP1. The highest degree of similarity occurs in the C-terminal region, which shows considerable homology to the cytoskeletal protein talin (186745). Northern blot analysis detected expression of a 5-kb HIP12 transcript in brain, heart, kidney, pancreas, and liver, but not in lung or placenta. In ES cell-derived neurons, both HIP1 and HIP12 are highly expressed and distributed throughout the cytoplasm and cell processes with enrichment within the cis-Golgi. In contrast to HIP1, which is toxic in cell culture, HIP12 does not confer toxicity in the same assay systems. HIP12 does not interact with huntingtin but can interact with HIP1, suggesting a potential interaction in vivo that may influence the function of each respective protein. By searching EST databases for homologs of yeast Sla2p, Engqvist-Goldstein et al. (1999) identified mouse and human cDNAs encoding HIP1R. The deduced human protein, which is 91% identical to the mouse sequence, is identical to the KIAA0655 protein reported by Ishikawa et al. (1998). It is also identical to the shorter sequence reported by Seki et al. (1998) except that it contains approximately 180 additional amino acids in it N terminus, including a conserved domain implicated in the endocytic function of Sla2p. HIP1R has 3 predicted coiled coils and a C-terminal talin-like domain, which Engqvist-Goldstein et al. (1999) confirmed binds F-actin

in vitro. Northern blot analysis revealed that mouse Hip1r is expressed ubiquitously, with reduced expression in skeletal muscle and heart, consistent with RT-PCR analysis of human HIP1R expression (Seki et al., 1998; Ishikawa et al., 1998). Fluorescence microscopy demonstrated that mouse Hip1r is expressed as punctate structures, enriched at the cell cortex and excluded from the nucleus, which colocalize with clathrin (see 118955) and other markers of receptor-mediated endocytosis.

Full details of the abovementioned studies are described in the following publications, the disclosure of which are hereby incorporated by reference:

Chopra, V. S.; Metzler, M.; Rasper, D. M.; Engqvist-Goldstein, A. E. Y.; Singaraja, R.; Gan, L.; Fichter, K. M.; McCutcheon, K.; Drubin, D.; Nicholson, D. W.; Hayden, M. R. : HIP12 is a non-proapoptotic member of a gene family including HIP1, an interacting protein with huntingtin. Mammalian Genome 11: 1006-1015, 2000. ; and

Engqvist-Goldstein, A. E. Y.; Kessels, M. M.; Chopra, V. S.; Hayden, M. R.; Drubin, D. G. : An actin-binding protein of the Sla2/Huntingtin interacting protein 1 family is a novel compo.

Further studies establishing the function and utilities of HIP12 are found in John Hopkins OMIM database record ID 605613, and in sited publications numbered 10160, 20936 and 20937-20938 listed in the bibliography section hereinbelow, which are also hereby incorporated by reference. .

Reference is now made to ZNF212 BINDING SITE. zinc finger protein 212 (ZNF212, Accession NM_012256) is a host target gene of VGAM1931, corresponding to VGAM1931-HOST TARGET GENE of Fig. 15A. ZNF212 BINDING SITE is a binding site found in the 3' untranslated region of ZNF212, corresponding to a host binding site such as BINDING SITE-I, BINDING SITE-II or BINDING SITE-III, all of Fig. 15A. Fig. 15D illustrates the complementarity of the nucleotide sequence of ZNF212 BINDING SITE, designated SEQ ID:14557, to the nucleotide sequence of VGAM1931 RNA of Fig. 15A, designated SEQ ID:4642.

A further function of VGAM1931 is therefore inhibition of zinc finger protein 212 (ZNF212). Accordingly, utilities of VGAM1931 include diagnosis and treatment of diseases and clinical conditions with which ZNF212 is associated. .

Reference is now made to FLJ20436 BINDING SITE. FLJ20436 (Accession NM_017822) is a host target gene of VGAM1931, corresponding to VGAM1931-HOST TARGET GENE of Fig. 15A. FLJ20436 BINDING SITE is a binding site found in the 3' untranslated region of FLJ20436, corresponding to a host binding site such as BINDING SITE-I, BINDING SITE-II or BINDING SITE-III, all of Fig. 15A. Fig. 15D illustrates the complementarity of the nucleotide sequence of FLJ20436 BINDING SITE, designated SEQ ID:19472, to the nucleotide sequence of VGAM1931 RNA of Fig. 15A, designated SEQ ID:4642.

Yet a further function of VGAM1931 is therefore inhibition of (FLJ20436). Accordingly, utilities of VGAM1931 include diagnosis and treatment of diseases and clinical conditions with which FLJ20436 is associated.

Reference is now made to KIAA1622 BINDING SITE. KIAA1622 (Accession NM_058237) is a host target gene of VGAM1931, corresponding to VGAM1931-HOST TARGET GENE of Fig. 15A. KIAA1622 BINDING SITE is a binding site found in the 3' untranslated region of KIAA1622, corresponding to a host binding site such as BINDING SITE-I, BINDING SITE-II or BINDING SITE-III, all of Fig. 15A. Fig. 15D illustrates the complementarity of the nucleotide sequence of KIAA1622 BINDING SITE, designated SEQ ID:27766, to the nucleotide sequence of VGAM1931 RNA of Fig. 15A, designated SEQ ID:4642.

Another function of VGAM1931 is therefore inhibition of (KIAA1622). Accordingly, utilities of VGAM1931 include diagnosis and treatment of diseases and clinical conditions with which KIAA1622 is associated.

Reference is now made to LOC51312 BINDING SITE. LOC51312 (Accession NM_018579) is a host target gene of VGAM1931, corresponding to VGAM1931-HOST TARGET GENE of Fig. 15A. LOC51312 BINDING SITE is a binding site found in the 5' untranslated region of LOC51312, corresponding to a host binding site such as BINDING SITE-I, BINDING SITE-II or BINDING SITE-III, all of Fig. 15A. Fig. 15D illustrates the complementarity of the nucleotide sequence of LOC51312 BINDING SITE, designated SEQ ID:20659, to the nucleotide sequence of VGAM1931 RNA of Fig. 15A, designated SEQ ID:4642.

Yet another function of VGAM1931 is therefore inhibition of (LOC51312). Accordingly, utilities of VGAM1931 include diagnosis and treatment of diseases and clinical conditions with which LOC51312 is associated.

Reference is now made to LOC57105 BINDING SITE. LOC57105 (Accession NM_020377) is a host target gene of VGAM1931, corresponding to VGAM1931-HOST TARGET GENE of Fig. 15A. LOC57105 BINDING SITE is a binding site found in the 3' untranslated region of LOC57105, corresponding to a host binding site such as BINDING SITE-I, BINDING SITE-II or BINDING SITE-III, all of Fig. 15A. Fig. 15D illustrates the complementarity of the nucleotide sequence of LOC57105 BINDING SITE, designated SEQ ID:21639, to the nucleotide sequence of VGAM1931 RNA of Fig. 15A, designated SEQ ID:4642.

An additional function of VGAM1931 is therefore inhibition of (LOC57105). Accordingly, utilities of VGAM1931 include diagnosis and treatment of diseases and clinical conditions with which LOC57105 is associated.

Reference is now made to LOC146603 BINDING SITE. LOC146603 (Accession XM_085514) is a host target gene of VGAM1931,

corresponding to VGAM1931-HOST TARGET GENE of Fig. 15A. LOC146603 BINDING SITE is a binding site found in the 5' untranslated region of LOC146603, corresponding to a host binding site such as BINDING SITE-I, BINDING SITE-II or BINDING SITE-III, all of Fig. 15A. Fig. 15D illustrates the complementarity of the nucleotide sequence of LOC146603 BINDING SITE, designated SEQ ID:38215, to the nucleotide sequence of VGAM1931 RNA of Fig. 15A, designated SEQ ID:4642.

A further function of VGAM1931 is therefore inhibition of (LOC146603). Accordingly, utilities of VGAM1931 include diagnosis and treatment of diseases and clinical conditions with which LOC146603 is associated.

Reference is now made to LOC145761 BINDING SITE. LOC145761 (Accession XM_096855) is a host target gene of VGAM1931, corresponding to VGAM1931-HOST TARGET GENE of Fig. 15A. LOC145761 BINDING SITE is a binding site found in the 5' untranslated region of LOC145761, corresponding to a host binding site such as BINDING SITE-I, BINDING SITE-II or BINDING SITE-III, all of Fig. 15A. Fig. 15D illustrates the complementarity of the nucleotide sequence of LOC145761 BINDING SITE, designated SEQ ID:40584, to the nucleotide sequence of VGAM1931 RNA of Fig. 15A, designated SEQ ID:4642.

Yet a further function of VGAM1931 is therefore inhibition of (LOC145761). Accordingly, utilities of VGAM1931 include diagnosis and treatment of diseases and clinical conditions with which LOC145761 is associated. .

Reference is now made to LOC202986 BINDING SITE. LOC202986 (Accession XM_117489) is a host target gene of VGAM1931, corresponding to VGAM1931-HOST TARGET GENE of Fig. 15A. LOC202986 BINDING SITE is a binding site found in the 3' untranslated region of LOC202986, corresponding to a host binding site such as BINDING SITE-I, BINDING SITE-II or BINDING SITE-III, all of Fig. 15A. Fig. 15D illustrates the complementarity of the nucleotide sequence of LOC202986 BINDING SITE, designated SEQ ID:43470, to the nucleotide sequence of VGAM1931 RNA of Fig. 15A, designated SEQ ID:4642.

Another function of VGAM1931 is therefore inhibition of (LOC202986). Accordingly, utilities of VGAM1931 include diagnosis and treatment of diseases and clinical conditions with which LOC202986 is associated.

Delete paragraphs [0162] to [57562].